Global urban temporal trends in fine particulate matter ($PM_{2·5}$) and attributable health burdens: estimates from global datasets

Veronica A Southerland, Michael Brauer, Arash Mohegh, Melanie S Hammer, Aaron van Donkelaar, Randall V Martin, Joshua S Apte, Susan C Anenberg

Summary

Background With much of the world's population residing in urban areas, an understanding of air pollution exposures at the city level can inform mitigation approaches. Previous studies of global urban air pollution have not considered trends in air pollutant concentrations nor corresponding attributable mortality burdens. We aimed to estimate trends in fine particulate matter ($PM_{2·5}$) concentrations and associated mortality for cities globally.

Methods We use high-resolution annual average $PM_{2·5}$ concentrations, epidemiologically derived concentration response functions, and country-level baseline disease rates to estimate population-weighted $PM_{2·5}$ concentrations and attributable cause-specific mortality in 13 160 urban centres between the years 2000 and 2019.

Findings Although regional averages of urban $PM_{2·5}$ concentrations decreased between the years 2000 and 2019, we found considerable heterogeneity in trends of $PM_{2·5}$ concentrations between urban areas. Approximately 86% (2·5 billion inhabitants) of urban inhabitants lived in urban areas that exceeded WHO's 2005 guideline annual average $PM_{2·5}$ (10 μg/m³), resulting in an excess of 1·8 million (95% CI 1·34 million–2·3 million) deaths in 2019. Regional averages of $PM_{2·5}$-attributable deaths increased in all regions except for Europe and the Americas, driven by changes in population numbers, age structures, and disease rates. In some cities, $PM_{2·5}$-attributable mortality increased despite decreases in $PM_{2·5}$ concentrations, resulting from shifting age distributions and rates of non-communicable disease.

Interpretation Our study showed that, between the years 2000 and 2019, most of the world's urban population lived in areas with unhealthy levels of $PM_{2·5}$, leading to substantial contributions to non-communicable disease burdens. Our results highlight that avoiding the large public health burden from urban $PM_{2·5}$ will require strategies that reduce exposure through emissions mitigation, as well as strategies that reduce vulnerability to $PM_{2·5}$ by improving overall public health.

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Introduction

Despite progress in reducing exposure in some countries, the global health burden of ambient fine particulate matter ($PM_{2·5}$) is increasing annually.[1,2] Long-term exposure to $PM_{2·5}$ is associated with premature mortality from a variety of diseases, including cardiovascular disease, respiratory disease, lung cancer, and lower respiratory infection.[3–5] $PM_{2·5}$ is now the leading environmental contributor to the global burden of disease, rising from being the fifth leading contributor among environmental risk factors in 1990, in part driven by declines in household air pollution and unsafe water and sanitation.[1] Recent estimates of $PM_{2·5}$-attributable disease burdens describe global, regional, and national trends, and do not focus on the city scale where 55% of the world's population resides; a figure that is expected to increase.[6] City-level decision makers and non-governmental organisations (eg, C40 Cities and ICLEI–Local Governments for Sustainability) can benefit from information on urban air pollution trends to drive policy change, analyse the effectiveness of environmental and air pollution mitigation policies, and track progress towards urban Sustainable Development Goals.

Assessing ambient $PM_{2·5}$-attributable mortality in cities globally is now possible owing to the availability of pollutant concentrations, population, urbanicity, disease rates, and epidemiological concentration-response relationships that have global coverage. Studies examining $PM_{2·5}$ concentrations in urban areas have typically focused on high $PM_{2·5}$ concentrations in megacities,[7,8] although a study by Joshua Apte and colleagues[9] considerably expands the knowledge of $PM_{2·5}$ exposures in urban areas by providing temporal estimates of concentrations in 4231 globally representative urban areas. Underscoring the need for $PM_{2·5}$ concentration estimates for a greater number of urban areas, their study found that the most polluted cities were those with less than 1 million inhabitants, most of which did not

Research in context

Evidence before this study

Fine particulate matter ($PM_{2·5}$) is considered the leading environmental health risk factor, associated with between 3 million and 4 million premature deaths worldwide each year, yet little is known about how $PM_{2·5}$-atttributable disease burdens compare across urban areas globally. We searched PubMed and Google Scholar between Sept 1, 2019, and June 15, 2021, for studies assessing the health risks of $PM_{2·5}$ in urban areas, limiting our search to articles published in English. We found a few studies that estimated urban $PM_{2·5}$-attributable health burdens but only for subsets of urban areas and only for a single year. Although several studies assessed a large number of cities in Europe and China, only one study in China analysed both a large number of urban areas for multiple years, although only for a limited number of year intervals.

Added value of this study

Our study expands on previous studies by estimating long-term trends in ambient $PM_{2·5}$ and associated mortality across 13 160 urban areas. We used globally consistent methods that are compatible with the 2019 Global Burden of Disease (GBD) study so that our results can be used in efforts relying on GBD to advance global public health. We found that the majority of urban inhabitants lived in cities where $PM_{2·5}$ levels exceeded the WHO guideline of 5 μg/m³ annual average concentration, and estimate that approximately a third of $PM_{2·5}$-attributable deaths could have been avoided had all cities met the WHO 2005 guideline of 10 μg/m³ (now the Interim Target 4) between the years 2000 and 2019. Further, we analysed the contributions of changing concentrations versus demographic shifts to $PM_{2·5}$-attributable mortality in urban areas and found baseline disease rates and demographic trends often counteracted decreases in $PM_{2·5}$ concentrations.

Implications of all the available evidence

Our results are consistent with previous studies that found global increases in $PM_{2·5}$ concentrations and attributable health burdens. Additionally, our study shows the importance of providing estimates at the city level owing to the wide heterogeneity of trends in $PM_{2·5}$ concentrations and mortality in urban areas. Our finding that demographic changes often counteract air quality improvements highlights the importance of a public health approach that both mitigates emissions and reduces vulnerability to $PM_{2·5}$ by improving overall public health. We also provide a dataset of $PM_{2·5}$ concentrations and attributable mortality for 13 160 cities that can be used to inform air quality management approaches at local, national, and regional scales.

have ground monitoring data for $PM_{2·5}$ concentrations. Their study also showed widening disparities (>50%) between urban exposures in high-income countries and low-income countries, noting improvements in $PM_{2·5}$ concentrations in high-income urban areas, while concentrations in lower-income urban areas increased.

Estimates of city-level $PM_{2·5}$-attributable health burdens have typically provided only single year estimates[10,11] for a small number of urban areas, although a growing number of health impact assessments estimate $PM_{2·5}$-attributable health burdens for subsets of urban areas in China[12–16] and Europe.[17] Estimating health impacts for 980 European cities in 2015, Khomenko and colleagues[17] showed the importance of estimating city-level $PM_{2·5}$-attributable health burdens by describing the heterogeneity of $PM_{2·5}$-attibutable mortality estimates in European cities. Of the other studies mentioned, only Zhu and colleagues[16] assessed both a large sample (n=129) of urban areas at three temporal intervals (2006, 2010, and 2015) using the 2010 Global Burden of Disease (GBD) study. Assessing temporality allowed Zhu and colleagues to examine inconsistencies between overall decreasing urban $PM_{2·5}$ concentrations and modest increases in $PM_{2·5}$-attributable mortality, owing to demographic changes, including increasing baseline mortality rates and an ageing population. In our study, we expected to find substantial heterogeneity in temporal trends in urban $PM_{2·5}$ concentrations and disease burdens, as well as drivers of those trends, in cities around the world.

In this study, we aimed to improve on these studies to estimate trends in $PM_{2·5}$ concentrations and associated mortality for cities globally by: (1) examining a larger subset of global cities (13 160 compared with tens or hundreds, and in one case thousands), (2) using finer scale $PM_{2·5}$ concentration estimates (approximately 1 km² compared with approximately 100 km²), (3) using methods compatible with the GBD 2019 study, (4) calculating temporal trends in concentrations and disease burdens for nearly two decades (2000 to 2019), and (5) estimating the number of $PM_{2·5}$-attributable deaths that would have been avoided if cities had met the WHO guideline for annual average $PM_{2·5}$. Resulting $PM_{2·5}$-attributable health impact estimates can inform air quality management approaches at local, national, and regional scales.

Methods

Population-weighted annual average concentrations

Although the GBD 2019 provides datasets for population and ambient $PM_{2·5}$ concentrations at a $0·1° \times 0·1°$ resolution, in our study we used fine resolution estimates at a $0·00833° \times 0·00833°$ (approximately 1 km²) resolution for both population and concentration estimates to better match the resolution of our urban spatial extent dataset. For $PM_{2·5}$ concentrations, we used a dataset that integrated information from satellite-retrieved aerosol optical depth, chemical transport modelling, and ground monitor data,[18] improving on previous model estimates[19] to provide surface-level $PM_{2·5}$ concentration estimates for the years 2000 to 2019. Briefly, multiple aerosol optical

depth retrievals from three satellite instruments (the Moderate Resolution Imaging Spectroradiometer, SeaWiFs, and the Multiangle Imaging Spectroradiometer) were combined and related to near-surface $PM_{2·5}$ concentrations using the Goddard Earth Observing System-Chem chemical transport model (appendix p 4). Ground-based observations of $PM_{2·5}$ were then incorporated using a geographically weighted regression. Estimated annual average concentrations were highly consistent with out-of-sample ground monitoring measurements (R^2=0·90–0·92). We compared urban $PM_{2·5}$ annual population-weighted concentrations that were calculated with both fine resolution $PM_{2·5}$ concentrations from the main analysis and more coarsely resolved $PM_{2·5}$ concentration estimates used in the GBD 2019 for national (and in some countries, subnational) scale analysis (appendix pp 3–4).

Gridded population count estimates were available from WorldPop for all ages for 2000 to 2019 at a 1 km² gridded resolution.[20,21] For urban area definitions, we used urban boundaries defined by the Global Human Settlement Grid for 13 160 urban areas,[22] also available at a 1 km² gridded resolution. Further description of these datasets are available in the appendix (p 2).

For each urban area, we divided the sum of the product of concentration estimates at the grid cell (k) by population at the grid cell (k) within each urban area (i), divided by the sum of population per grid cells (k) of each urban area (i).

$$\sum(\text{population}_{i,k} \times \text{concentration}_{i,k}) / \sum \text{population}_{i,k}$$

Health impact function

We used a health impact function to estimate mortality attributable to $PM_{2·5}$, following previous studies.[23–26] The health impact function incorporated annual average $PM_{2·5}$ concentrations, population counts, baseline mortality rates, and epidemiologically derived concentration response functions relating $PM_{2·5}$ concentrations and health outcomes. The population-attributable fraction describes the percentage of disease in a given population that is attributable to $PM_{2·5}$ on the basis of concentration response functions derived from the epidemiological literature. The population attributable fraction ($PAF_{h,i,a}$) incorporated the relative risk (RR) derived from the epidemiological literature, calculated for each urban area (i), age group (a), and cause-specific mortality endpoint (h).

$$PAF_{h,i,a} = (RR_{h,i,a} - 1) / RR_{h,i,a}$$

We then calculated the total $PM_{2·5}$-attributable mortality burden using the following equation:

$$y_{h,i,a} = m_{h,i,a} \times p_{i,a} \times PAF_{h,i,a}$$

in which y indicates the number of cases of the health outcome (h) attributable to $PM_{2·5}$ per city (i) and age group (a); p represents the population count for each city (i) and age group (a); and m indicates the baseline disease rate for each city (i) using country-level baseline disease rates, for each health endpoint (h) and age group (a).

We used cause-specific RR estimates from the GBD 2019 study for mortality from ischaemic heart disease, ischaemic and intracerebral haemorrhagic stroke, lower respiratory infections, lung cancer, type 2 diabetes, and chronic obstructive pulmonary disease. We applied RR estimates for inhabitants aged between 25 and 99 years in 5 year increments for ischaemic heart disease and ischaemic and intracerebral haemorrhagic stroke. The GBD project also did a meta-analysis and regression of available epidemiological studies, resulting in 1000 splined meta-regression estimates for 385 integer exposure levels ranging from 0 μg/m³ to 2500 μg/m³.[27] To convert these regression estimates to RR estimates for use in our health impact function (appendix p 1), we applied the same theoretical minimum risk exposure level used in the GBD 2019, assuming a uniform distribution between 2·4 μg/m³ and 5·9 μg/m³. We present uncertainty in attributable mortality estimates from the health impact function by estimating results at the 2.5th and 97.5th percentile of the RR estimate. Cause-specific $PM_{2·5}$-attributable mortality estimates were summed to yield total $PM_{2·5}$-attributable mortality.

See Online for appendix

We obtained country-specific, age-specific, and cause-specific baseline disease rates from the 2019 GBD study data exchange for 2000 to 2019. In the absence of a global scale dataset on urban mortality rates, we applied these national rates to the urban areas used in our study (figure 1A). We estimated the fraction of the population in each age group by back-calculating population fractions from the GBD data for number of deaths and death rate per 100 000 people.

For more on GHDx tool see http://ghdx.healthdata.org/gbd-results-tool

As concentrations, baseline disease rates, and population growth and ageing all vary over time, we assessed the proportional contribution of each of these factors to the change in $PM_{2·5}$-attributable mortality. Additional information regarding the theoretical minimum risk exposure level, RR calculation, and proportional contribution analysis is provided in the appendix (p 1). All calculations and data visualisation for this analysis were done in R, version 3.5.3.

Role of the funding source

The funders of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

For the year 2019, we found that the mean population-weighted ambient $PM_{2·5}$ concentration was 35 μg/m³ (SD 26 μg/m³) across all urban areas globally, which was the same as for the year 2000 (35 μg/m³, SD 25 μg/m³). However, this concentration was still

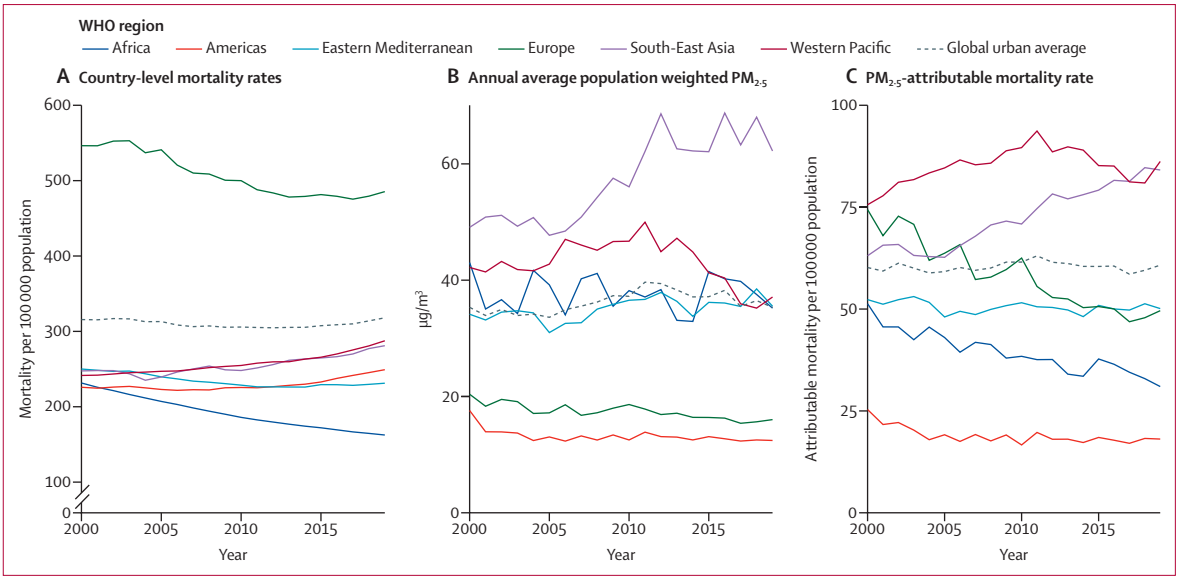

Figure 1: **Trends in global urban mortality rates and $PM_{2·5}$ concentrations, 2000 to 2019**
(A) Country-level baseline mortality. (B) Regional urban averages of population-weighted annual average $PM_{2·5}$ concentrations. (C) Regional urban averages of $PM_{2·5}$-attributable mortality rates.

over seven times the 2021 WHO guideline for annual average $PM_{2·5}$ (5 µg/m³) and over three times the 2005 guideline level, which is now Interim Target 4 (10 µg/m³). Despite consistency in global urban annual average $PM_{2·5}$ concentrations across time, concentration trends from 2000 to 2019 varied widely between regions (figure 1B). The region with the largest absolute decrease in annual average urban concentrations was Africa, where region-wide average concentrations decreased by 18% (from 43 µg/m³ in 2000 to 35 µg/m³ in 2019), although we observed a large degree of intra-regional variation (mean for the net difference in concentration between 2019 and 2000 for all cities in the region: –7 ug/m³, SD of difference 10 ug/m³). Similarly, in urban areas in southeast Asian countries (including India) where regional average increases were largest, the mean urban population-weighted concentration increased by 27% (from 49 µg/m³ to 62 µg/m³), although trends also varied substantially between urban areas (SD 26 µg/m³ to 28 µg/m³). Urban $PM_{2·5}$ concentration trends varied substantially in the Americas (mean difference –5 µg/m³, SD of difference 9 µg/m³) and in the western Pacific (including China); mean difference –3 µg/m³, SD of difference 20 µg/m³; figure 2). We estimated that approximately 85% of urban inhabitants globally lived in urban areas exceeding the 2005 WHO guideline (10 µg/m³) in both 2000 (1·99 billion people) and 2019 (2·5 billion). Only 16% (n=2054) of all urban areas globally ever met the 2005 WHO guideline between 2000 and 2019. Most cities that ever attained the 2005 WHO guideline were located in the Americas (n=988; 48%), Africa (n=347; 17%), and Europe (n=269; 13%).

We next incorporated baseline disease rates to estimate $PM_{2·5}$-attributable mortality in urban areas. The global average urban $PM_{2·5}$-attributable mortality rate was 61 (95% CI 45–77) deaths per 100 000 inhabitants in 2019. Regional averages for 2019 in the Americas (18 [10–28]) and Africa (31 [22–40]) were below the global urban average, while urban averages in the Western Pacific (86 [66–107]) and South-East Asia (84 [66–101]) were above the global urban average (figure 1C). Moreover, urban averages in Europe (50 [32–70]) and the Eastern Mediterranean (50 [37–63]) were near the global urban average. Of all regions globally, cities in South-East Asia had the largest increase in $PM_{2·5}$-attributable mortality rates over this time period (33%; 63 to 84 per 100 000), followed by cities in the western Pacific (14%; 76 to 86). Cities in Africa had the largest decrease (–40%; 51 to 31), followed by cities in Europe (–33%; 74 to 50) and cities in the Americas (–29%, 25 to 18).

Attributable mortality among the top 250 most populated cities comprised 43% of the total global attributable cases in the year 2000; 563 000 (95% CI 417 000–717 000) of 1·3 million cases (970 000–1·67 million) and 47% of the total global attributable cases in 2019 (837 000 [627 000–1·06 million] of 1·8 million [1·34 million–2·3 million] total deaths in urban areas). This contribution was proportional to the percentage of the population living in urban areas globally (990 million [45%] of 2·2 billion in the year 2000, and 1·36 billion [47%] of 2·9 billion in the year 2019). Attributable mortality estimates for the top 250 most populated cities and results for all 13 160 urban areas are available in the appendix (pp 6–15).

In many urban areas, directional trends in $PM_{2·5}$ concentrations did not correspond with trends in

$PM_{2·5}$-attributable mortality rates. We highlighted two examples of the differences between trends in $PM_{2·5}$ population-weighted concentrations (figure 3A, B) and $PM_{2·5}$-attributable mortality (figure 3C, D). While Guangzhou (China) had a decrease in population-weighted $PM_{2·5}$ (–14%; 37 μg/m³ to 32 μg/m³), $PM_{2·5}$-attributable mortality rates increased (+10%; 82 to 90 per 100 000). Contrastingly, Luanda (Angola) had an increase in $PM_{2·5}$ population-weighted concentrations (+38%; 13 μg/m³ to 18 μg/m³), but a decrease in $PM_{2·5}$-attributable mortality rates (–16%; 19 μg/m³ to 16 μg/m³).

We also estimated $PM_{2·5}$-attributable mortality for a scenario under which urban concentrations were reduced to the 2005 WHO guideline of 10 μg/m³ per annual average (now Interim Target 4, as the guideline was changed to 5 μg/m³ in 2021). We estimated that over 1·21 million (95% CI 984 000–1·34 million) deaths in urban areas globally could have been avoided in 2019 if all urban areas had met WHO's guideline (1·8 million [1·34 million–2·3 million] at 2019 values versus 590 000 [361 000–943 000] deaths had all urban areas met the WHO guideline). Between 2000 and 2019, over 30·5 million (22·8–38·7) deaths attributable to $PM_{2·5}$ are estimated to have occurred in urban areas, with over 9·6 million (5·9 million–15·7 million; 32% [26%–41%]) of those considered to be avoidable had all urban areas met the WHO guideline during the entire 19 year time period.

We examined the contribution of each health impact function parameter to the overall change in $PM_{2·5}$-attributable mortality between the years 2000 and 2019. We found that changes in population growth and population ageing were the largest drivers of total $PM_{2·5}$-attributable deaths in all regions (figure 4). Population ageing increased $PM_{2·5}$-attributable mortality in all regions apart from Europe and the Americas. Changes in baseline disease rates had more impact on $PM_{2·5}$-attributable mortality than did $PM_{2·5}$ concentrations in cities in Africa, the Eastern Mediterranean, and South-East Asia. The opposite was true for the Americas, Europe, and the Western Pacific, where decreases in $PM_{2·5}$ concentrations outweighed the impact of baseline disease rates.

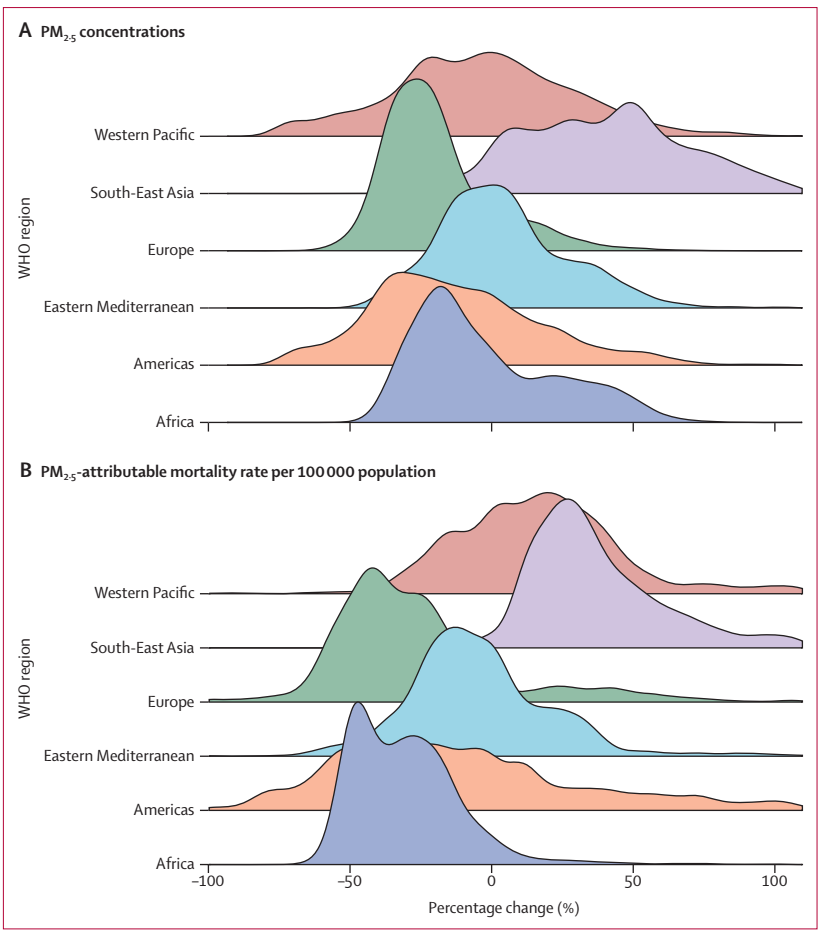

Figure 2: **Density distribution of percent change between 2000 and 2019 for all urban areas**
Percentage change among all urban areas (n=13 160) for population-weighted $PM_{2·5}$ concentrations (A) and $PM_{2·5}$-attributable mortality per 100 000 population (B).

Discussion

In this study, we found that the global average urban $PM_{2·5}$ concentration in 2019 was 35 μg/m³, which is over three times the WHO 2005 guideline for annual average $PM_{2·5}$ (10 μg/m³), resulting in 45 to 77 (95% CI) premature deaths per 100 000 people. In 2019, approximately 86% (2·5 billion inhabitants) of urban inhabitants lived in areas that exceeded WHO's 2005 guideline, resulting in an excess of 1·8 (95% CI 1·34–2·3) million deaths and accounting for 43% of the 4·14 million global ambient $PM_{2·5}$-attributable deaths in 2019 estimated by the GBD 2019. Population-weighted $PM_{2·5}$ concentrations and attributable mortality were relatively unchanged between 2000 and 2019 across all urban areas globally, although global trends mask considerable regional variation. The largest global increase in annual average $PM_{2·5}$ concentrations and $PM_{2·5}$-attributable mortality per 100 000 inhabitants occurred in cities in South-East Asia (27%), including cities in India (33%).

Our results are consistent with estimates from Apte and colleagues[9] of $PM_{2·5}$ trends in urban areas, whereby mean regional estimates were within approximately 4% agreement for 2018. Although both analyses use globally gridded estimates of annual average $PM_{2·5}$ concentrations provided by Hammer and colleagues,[18] there were several methodological differences with our study. We used a gridded definition of urban areas,[22] which was inclusive of urban areas (n=13 160) that had more than 50 000 inhabitants. Apte and colleagues apply circular buffers to city centroids using the Universe of Cities dataset, which was inclusive of urban areas (n=4321) that had more than 100 000 inhabitants;[28] however, Apte and colleagues found minimal difference in results that were estimated using a circular buffer versus area-weighted

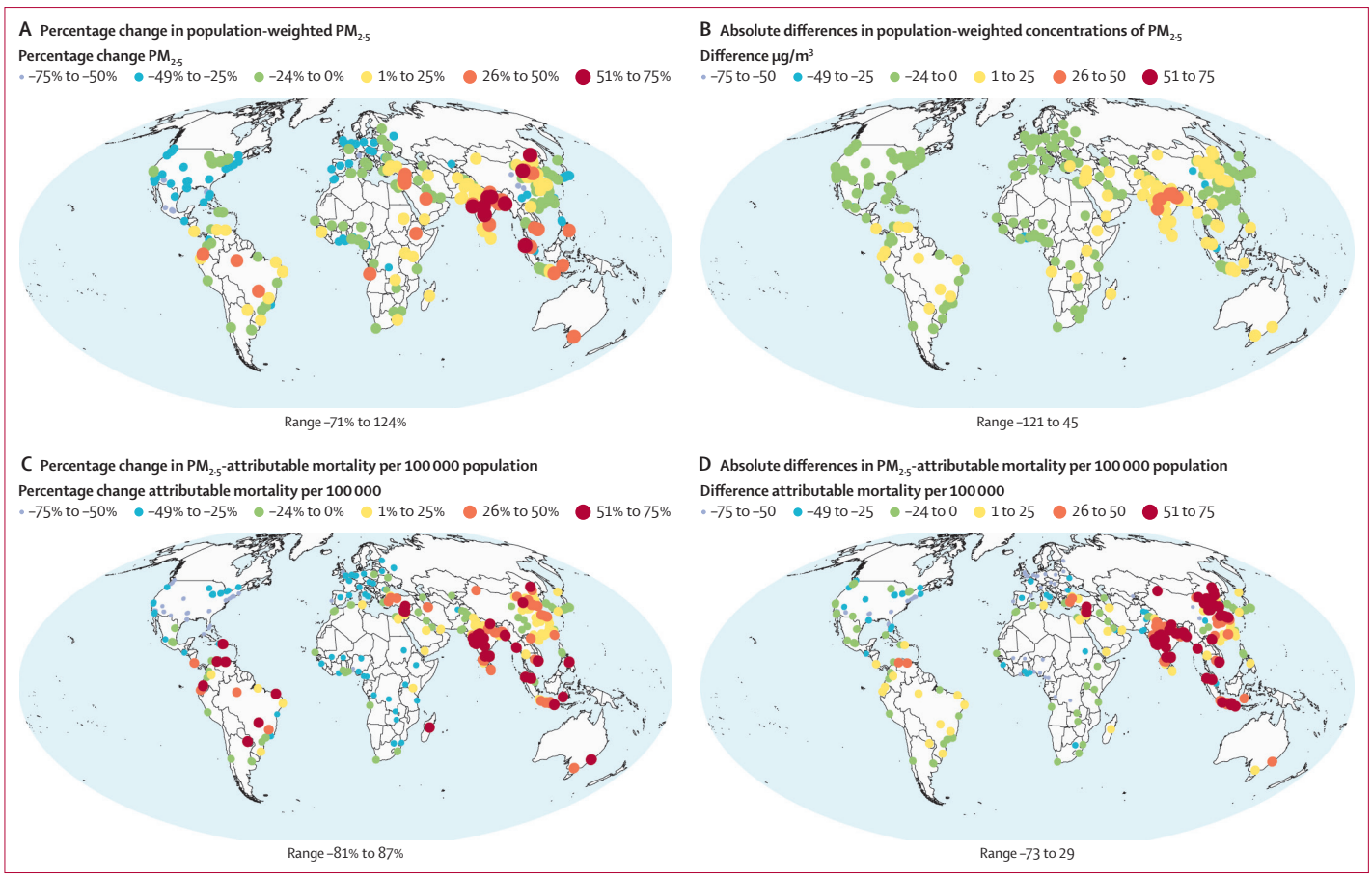

Figure 3: **Change in population-weighted $PM_{2·5}$ concentrations and $PM_{2·5}$-attributable mortality rates between 2000 and 2019 for the top 250 most populated urban areas based on 2019 WorldPop estimates**

(A) Percentage change in population-weighted $PM_{2·5}$ concentrations. (B) Absolute differences in population-weighted concentrations of $PM_{2·5}$. (C) Percentage change in $PM_{2·5}$-attributable mortality per 100 000 population. (D) Absolute differences in $PM_{2·5}$-attributable mortality per 100 000 population.

concentrations. These differences meant that we included many more cities with a smaller average population. Moreover, we incorporated gridded population estimates from WorldPop while Apte and colleagues scale population estimates from 2010[28] using decadal average population growth rates for years before 2010, and population growth rates from the UN World Urbanisation Prospects database for years after 2010.[6] Jointly, these differences in gridded population estimates and urban boundary definitions accounted for the slightly higher global urban population-weighted average $PM_{2·5}$ concentration estimates reported in our study (37 µg/m³ *vs* 31 µg/m³ in 2018), as well as potential differences in estimates provided here and previously published city-level $PM_{2·5}$ and population estimates. The difference in concentration data source and averaging approach lead to discrepancies between our work and other reported urban concentrations. Our study also went a step further by applying the $PM_{2·5}$ concentrations to estimate $PM_{2·5}$-attributable mortality trends.

By incorporating temporal trends and finer spatial resolution data for population and concentration input datasets, we improved on previous $PM_{2·5}$ health impact assessments in urban areas that used a coarser spatial resolution and reported estimates for only a single year.[11,12,14,16,17] Coarse resolution concentration datasets could dilute high urban concentrations,[29] particularly when concentrations overlap with densely populated areas.[30] Although one study found only a small impact of grid resolution on $PM_{2·5}$-attributable health impacts,[31] most studies reported that grid resolution substantially influenced results, with coarser resolutions leading to $PM_{2·5}$ concentration underestimates.[32–34]

We found that decreasing $PM_{2·5}$ concentrations in urban areas did not necessarily correspond with decreases in estimated $PM_{2·5}$-attributable mortality rates, showing that demographic factors are influential drivers of estimated $PM_{2·5}$-attributable mortality burden. Previous analyses of the global burden of $PM_{2·5}$ also reported that changes in mortality rates and age distributions often outweigh changes in air pollution exposure.[2,35] Mortality rates provided by the GBD 2019 account for risk factors and other exposures that influence baseline mortality, such as diet, rates of smoking, and access to health care. Further, we found that, compared with global averages,

decreases in $PM_{2.5}$ concentrations had a larger impact on total $PM_{2.5}$-attributable mortality in urban areas where concentrations are low (ie, the Americas and Europe). For example, in the relatively polluted Western Pacific (including China), urban $PM_{2.5}$ concentrations decreased by 12% between 2000 and 2019 (42 µg/m³ to 37 µg/m³), but $PM_{2.5}$-attributable deaths increased by 44% (525 400 to 754 000). Contrastingly, in European cities where concentrations are relatively low, the 20% reduction in concentrations (20 µg/m³ to 16 µg/m³) outweighed the contribution of a more vulnerable ageing population, resulting in a 23% decrease (237 600 to 182 000) in $PM_{2.5}$-attributable deaths. Although Europe also had a total decrease in population, this trend is in part due to the shape of the dose–response curve. At higher concentrations, where the dose–response curve is flatter, decreases in concentrations have comparatively less influence on $PM_{2.5}$-attributable deaths than decreases at lower concentrations where the dose–response curve is steeper. As mortality rates for health outcomes that are associated with air pollution are multifactorial and are driven by other factors in addition to air pollution levels, these findings indicate that a combination of more substantial air quality improvements and improved baseline health are needed to reduce the air pollution-related mortality.

Our study has several limitations, including our inability to fully account for uncertainties. Uncertainty was inherent in the estimates for each input in the health impact function,[36] including the RR estimates, population estimates, $PM_{2.5}$ concentration estimates, and baseline disease rates. Baseline disease rates were uncertain, one of the reasons being because we applied country-level baseline disease rates and age group compositions to urban areas, although urban baseline disease rates and demographics could differ from country-level averages.[37] Apte and colleagues[35] found only a 5% to 10% variation in regional aggregated totals when apportioning per-capita mortality rates for each region from rural to urban areas using a constant regional average. To further assess potential bias introduced by this approach, we compared urban-level to country-level cause-specific baseline mortality rates available from the GBD 2018 (n=97), finding no discernable pattern to suggest that using national rates systematically biases results in either direction (appendix p 5). Additionally, we did not account for the small fraction of the change in baseline disease rates that were dependent on $PM_{2.5}$. Finally, in this study, we only assessed $PM_{2.5}$ impacts on mortality burdens, which underestimates the full $PM_{2.5}$ health burden as $PM_{2.5}$ is also linked with low birthweight,[38] preterm birth,[39] and cognitive impairment.[40]

Despite these uncertainties, our study shows that while $PM_{2.5}$ concentrations and associated mortality burdens have declined in some parts of the world, $PM_{2.5}$ remains an important public health risk factor in urban areas worldwide. Understanding the factors that drive temporal trends in $PM_{2.5}$ concentrations and attributable mortality estimates can inform decision making aimed at reducing air pollution-related health burdens. Reducing the urban health burden attributable to $PM_{2.5}$ will require reducing exposures through emissions mitigation and by improving overall public health.

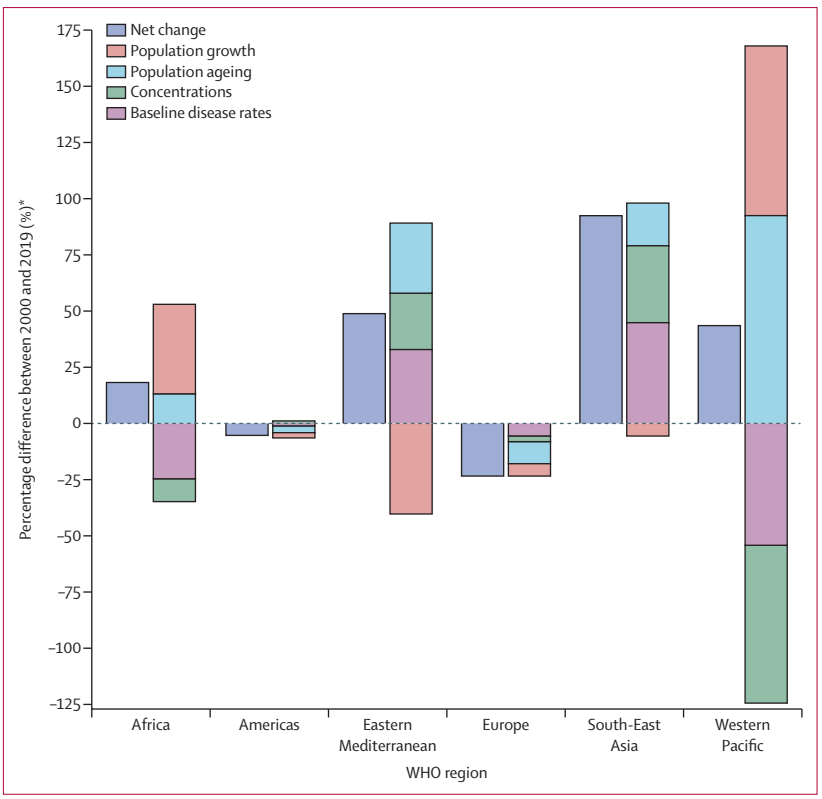

Figure 4: **Percent contribution of each health impact function to the change in $PM_{2.5}$-attributable mortality from 2000 to 2019 for all urban areas across regions.**
*Calculated using the methods described in the appendix (pp 2–3).

Contributors
SCA, VAS, MB, and JSA conceived the project idea. VAS and AM did the analysis. MSH, AvD, and RVM contributed to the development and verification of the fine particulate matter dataset used in this analysis. VAS had primary responsibility for writing the manuscript, to which all authors contributed.

Declaration of interests
We declare no competing interests.

Data sharing
Baseline disease rates are available from http://ghdx.healthdata.org/gbd-results-tool. WorldPop datasets are available at https://www.worldpop.org/geodata/listing?id=64. $PM_{2.5}$ concentration datasets are available at https://sites.wustl.edu/acag/datasets/surface-pm2-5/. Other input datasets are available upon request from VAS (vtinney@gwu.edu). Cause-specific mortality estimates are available upon request from VAS. The estimated urban $PM_{2.5}$-attributable concentrations and mortality results are available at: https://blogs.gwu.edu/sanenberg/.

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